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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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EXAMINER	
BUNNER, BRIDGET E	

ART UNIT	PAPER NUMBER
1647	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/693,480	ITESCU, SILVIU	
	Examiner	Art Unit	
	Bridget E. Bunner	1647	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *06 August 2009*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *35,37,43,46,47,49-51 and 57* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *35,37,43,46,47,49-51 and 57* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *01 February 2008 and 23 October 2003* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *5/27/09; 8/6/09*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Continued Examination Under 37 CFR 1.114

A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 06 August 2009 has been entered.

Status of Application, Amendments and/or Claims

Claims 35, 37, 43, 46, 47, 49-51 and 57 are under consideration in the instant application.

Double Patenting

1. Claims 35, 37, 43, 46-47, 49, 50, 51 and 57 are provisionally rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 69, 77-78, 82-84 of copending Application No. 11/234,879. Although the conflicting claims are not identical, they are not patentably distinct from each other because both sets of claims are directed to a method of treating a disorder comprising administering stromal-derived factor-1. This is a provisional obviousness-type double patenting rejection because the conflicting claims have not in fact been patented. The basis for this provisional rejection is set forth at pages 2-3 of the previous Office Action (03 February 2009), page 4 of the Office Action of 09 May 2008 and pages 6-7 of the Office Action of 08 August 2007.

At page 3 of the Response of 06 August 2009, Applicant maintains the position that the current rejection is provisional as the cited application is not patented or allowed. Applicant indicates consideration of the filing of a terminal disclaimer, if necessary, in the current application should the claims otherwise be deemed allowable and the rejection becomes non-provisional.

The rejection is maintained and held in abeyance until all other issues are resolved. It is noted that a Notice of Allowance in the '879 application was mailed on 09 July 2009. Applicant is encouraged to submit a terminal disclaimer at Applicant's earliest convenience.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

2. Claims 35, 37, 43, 46, 57 are rejected under 35 U.S.C. 103(a) as being unpatentable over Peterson, BE (US 2002/0094327; priority to 05 November 2000) in view of Hung et al. (US 2003/0171294; priority to 13 August 1999). The basis of the rejection is set forth at pages 3-7 of the previous Office Action (03 February 2009).

Applicant's arguments (06 August 2009), as they pertain to the previous rejection of record have been fully considered but are not deemed to be persuasive for the following reasons.

(i) At page 4 of the Response, Applicant argues that the Examiner has mischaracterized the claimed invention, which recites administering SDF-1 to induce regeneration of endogenous cardiomyocytes (i.e., by preventing apoptosis of cardiomyocytes and/or inducing proliferation of endogenous cardiomyocytes). Applicant indicates that claim 35 recites administering SDF-1 in such a manner that it acts on endogenous cardiomyocytes and that there is no suggestion in Peterson of this. Applicant argues that whether the SDF-1 may or may not additionally act on pluripotent stem cells, as suggested by the Examiner, is not relevant to the claimed method. Applicant adds that Hung et al. in combination with Peterson does not cure this deficiency. Applicant reiterates that the effect on endogenous cardiomyocytes is not expected or suggested in the prior art.

Applicant's arguments have been fully considered but are not found to be persuasive. Specifically, as discussed in the previous Office Action, Peterson teaches that a proposed alternative method for treating organ failure is organ regeneration wherein damaged cells of a failing organ are replaced with new, undamaged cells (page 1, [0004]). Peterson discloses that

the invention in the application relates to a method for selectively directing migration of pluripotent stem cells to a target tissue within a subject by modulating the level of SDF-1α protein in the target tissue (page 1, [0006]). Peterson continues to state that by "increasing the number of pluripotent stem cells that traffic to the target tissue, the rate of tissue repair can be increased because there will be a greater number of pluripotent stem cells in the target tissue that can differentiate into cells which can repopulate and partially or wholly restore the normal function of the damaged tissue" (page 1, [0006]). Peterson also teaches that the heart is one of the target tissues within a mammalian subject in which SDF-1α is administered (page 8, column 2, [0063]). Thus, in view of Peterson and Hung et al., it is clear that if a mammalian subject has SDF-1α injected into heart tissue, that subject has a damaged heart requiring cell regeneration, meeting the limitations of claim 35. Also, the disclosure of Peterson does not teach away from the instant claims because administration of SDF-1α to a target tissue (such as the heart) increases the number of pluripotent stem cells (that are already endogenous to the subject) that traffic to the tissue and differentiate into endogenous cells of that damaged tissue.

Additionally, since Peterson teaches the administration of SDF-1 to the same subject population and to the same tissue as recited in the claims, the regeneration of endogenous cardiomyocytes must have been inherently occurring in the prior art. The disclosure of Peterson fully meets the terms of the claimed method because SDF-1α inherently possesses endogenous cardiomyocyte regeneration activity, absent evidence to the contrary (*In re Best*, 562 F.2d 1252, 1255, 195 USPQ 430, 433 (CCPA 1977)). A compound and all of its properties are inseparable; they are one and the same thing and simply stating a new property of SDF-1α does not render the claimed method of the instant application free of the art (see *In re Papesch*, CCPA 137 USPQ

43; *In re Swinehart and Sfiligoj*, 169 USPQ 226 (CCPA 1971); *In re May*, 574 F.2d 1082, 1090, 197 USPQ 601, 607 (CCPA 1978)). Furthermore, inherent anticipation does not require that one of ordinary skill in the art recognize an inherent feature in a prior art disclosure (*Schering Corp. v. Geneva Pharmaceuticals Inc.*, 67 USPQ2d 1664 (CAFC 2003); *Toro Co. v. Deere & Co.*, 355 F.3d 1313, 1320, 69 USPQ2d 1584, 1590 (Fed. Cir. 2004)).

(ii) At the bottom of page 5 of the Response, Applicant asserts that for inherency to be established the claimed method must be a necessary consequence of following the prior art and not just a possible or even probable outcome. Applicant argues that the teachings of Peterson cannot necessarily result in the claimed method because the citation does not teach intramyocardial or intracoronary administration of SDF-1. Applicant indicates that the SDF-1 could be administered to any site in the body, or even a different site in the heart. At the top of page 6, Applicant states that there is no indication that this would induce regeneration of endogenous cardiomyocytes. Applicant adds that Hung et al. in combination does not cure this.

Applicant's arguments have been fully considered but are not found to be persuasive. Peterson teaches that the heart is one of the target tissues within a mammalian subject in which SDF-1α is administered (page 8, column 2, [0063]). Peterson also discloses that an SDF-1α composition of the invention may be administered to a mammalian subject neat or in pharmaceutically acceptable carriers in a manner selected on the basis of mode and route of administration and standard pharmaceutical practice (page 8, [0065]). Furthermore, Peterson gives a potential mechanistic example of the function of SDF-1α once administered to a tissue. At page 1, [0006], Peterson states:

"For example, where a liver has been damaged to the point where its hepatocytes cannot replicate in sufficient quantity to restore normal liver function, the levels of SDF-1 alpha protein can be artificially increased (e.g., by intrahepatic injection of the chemokine). The high local concentrations of SDF-1 alpha protein will then cause pluripotent stem cells (e.g., oval cells derived from hematopoietic stem cells) to be recruited and/or retained into the damaged liver at a greater than normal rate. Once in the liver, these pluripotent stem cells can differentiate (with or without the help of other agents such as morphogens) into new hepatocytes to replace the damaged cells and restore liver function."

Thus, Peterson clearly teaches the local administration of SDF-1α to damaged tissues, including the heart. Although Peterson does not specifically disclose intramyocardial or intracoronary administration of SDF-1α, Hung et al. teaches that it is routine to administer factors, such as angiogenic factors, via intramyocardial and intracoronary administration to the heart. Thus, it would have been obvious to the person of ordinary skill in the art at the time the invention was made to modify the method of administering of SDF-1α to heart tissue as taught by Peterson by utilizing intramyocardial or intracoronary administration as taught by Hung et al. The person of ordinary skill in the art would have been motivated to make that modification in order to localize cell migration/differentiation and tissue repair (see for example, Hung et al. page 1, [0007]; Peterson, page 1 [0004, 0006]). The person of ordinary skill in the art reasonably would have expected success because similar proteins and agents were already being intramyocardially and intracoronarily administered to the heart at the time the invention was made (see Hung et al.).

(iii) At the middle of page 6 of the Response of 06 August 2009, Applicant argues that inherent properties of SDF-1 cannot be used to support an argument as to why documents would be combined and also, the suggestion or motivation to combine or modify references must be

present prior to an applicant's date of invention. A previously unknown inherent property cannot supply this suggestion at the required time.

Applicant's arguments have been fully considered but are not found to be persuasive. Contrary to Applicant's assertion, inherent properties of SDF-1 were not used as support for the combination of Peterson and Hung et al. Additionally, the suggestion or motivation to combine or modify the references was present prior to the filing date of the instant application. As discussed in point (ii) above, Peterson does not specifically disclose intramyocardial or intracoronary administration of SDF-1α. However, Hung et al. teaches that it is routine to administer factors, such as angiogenic factors, via intramyocardial and intracoronary administration to the heart. Thus, it would have been obvious to the person of ordinary skill in the art at the time the invention was made to modify the method of administering of SDF-1α to heart tissue as taught by Peterson by utilizing intramyocardial or intracoronary administration as taught by Hung et al. The person of ordinary skill in the art would have been motivated to make that modification in order to localize cell migration/differentiation and tissue repair (see for example, Hung et al. page 1, [0007]; Peterson, page 1 [0004, 0006]). The person of ordinary skill in the art reasonably would have expected success because similar proteins and agents were already being intramyocardially and intracoronarily administered to the heart at the time the invention was made (see Hung et al.).

(iv) At page 6 of the Response, Applicant asserts that Peterson merely suggests administering SDF-1 to a tissue (not a specific site within a tissue, let alone the myocardium or the coronary circulation) to cause non-resident stem cells to migrate to that tissue. Applicant contends that

Peterson teaches that SDF-1 acts on non-resident stem cells, not on cardiomyocytes. Applicant indicates that Peterson provides no motivation or teaching to specifically administer SDF-1 to the myocardium or coronary circulation in such a manner that it induces regeneration of endogenous cardiomyocytes.

Applicant's arguments have been fully considered but are not found to be persuasive. Peterson discloses that the invention relates to a method for selectively directing migration of pluripotent stem cells to a target tissue within a subject by modulating the level of SDF-1α protein in the target tissue (page 1, [0006]). Peterson continues to state that by "increasing the number of pluripotent stem cells that traffic to the target tissue, the rate of tissue repair can be increased because there will be a greater number of pluripotent stem cells in the target tissue that can differentiate into cells which can repopulate and partially or wholly restore the normal function of the damaged tissue" (page 1, [0006]). Peterson also teaches that the heart is one of the target tissues within a mammalian subject in which SDF-1α is administered (page 8, column 2, [0063]). The disclosure of Peterson does not teach away from the instant claims because local administration of SDF-1α to a target tissue (such as the heart) increases the number of pluripotent stem cells (that are already endogenous to the subject) that traffic to the tissue and differentiate into endogenous cells of that damaged tissue. MPEP 2111(I) states that "the discovery of a previously unappreciated property of a prior art composition, or of a scientific explanation for the prior art's functioning, does not render the old composition patentably new to the discoverer." *Atlas Powder Co. v. Ireco Inc.*, 190 F.3d 1342,

1347, 51 USPQ2d 1943, 1947 (Fed. Cir. 1999). Additionally, the claiming of a new use, new function or unknown property which is inherently present in the prior art does not necessarily make the claim patentable (*In re Best*, 562 F.2d 1252, 1254, 195 USPQ 430, 433 (CCPA 1977)).

(v) At the top of page 7 of the Response, Applicant argues that Hung et al. fails to discuss any compound that is administered myocardially or intracoronarily that induces regeneration of endogenous cardiomyocytes (as opposed to blood vessels). Applicant asserts that Hung et al. only discusses using a growth factor to induce angiogenesis and does not contemplate SDF-1. Applicant adds that there is no reason to combine the teachings of Hung et al. to induce angiogenesis with growth factors with those of Peterson regarding attracting non-resident stem cells to a tissue.

Applicant's arguments have been fully considered but are not found to be persuasive. Peterson teaches that SDF-1α can be locally introduced into target tissues, such as the heart, in a mammalian subject (page 8, column 2, [0063]). Hung et al. simply discloses the successful intramyocardial and intracoronary administration of therapeutic proteins, such as FGF, to subjects. Thus, it would have been obvious to the person of ordinary skill in the art at the time the invention was made to modify the method of administering of SDF-1α to heart tissue as taught by Peterson by utilizing intramyocardial or intracoronary administration as taught by Hung et al. The person of ordinary skill in the art would have been motivated to make that modification in order to localize cell migration/differentiation and tissue repair (see for example, Hung et al. page 1, [0007]; Peterson, page 1 [0004, 0006]). The person of ordinary skill in the art reasonably would have expected success because similar proteins and agents were already

being intramyocardially and intracoronarily administered to the heart at the time the invention was made (see Hung et al.). Therefore, the claimed invention as a whole was clearly *prima facie* obvious over the prior art.

(vi) At the bottom of page 7 of the Response, Applicant asserts that the present method provides an unexpected result when considered in view of Peterson and Hung et al. Applicant indicates that the application exemplifies that cardiomyocytes are protected from apoptosis by SDF-1 and that SDF-1 induces endogenous cardiomyocyte proliferation/regeneration.

Applicant's arguments have been fully considered but are not found to be persuasive. Specifically, to summarize the teachings above, Peterson discloses the local administration of SDF-1α to damaged heart tissue. Hung et al. simply teaches that is routine to administer factors, such as angiogenic factors, via intramyocardial and intracoronary administration to the heart. MPEP 2111(I) states that "the discovery of a previously unappreciated property of a prior art composition, or of a scientific explanation for the prior art's functioning, does not render the old composition patentably new to the discoverer." *Atlas Powder Co. v. Ireco Inc.*, 190 F.3d 1342, 1347, 51 USPQ2d 1943, 1947 (Fed. Cir. 1999). Additionally, the claiming of a new use, new function or unknown property which is inherently present in the prior art does not necessarily make the claim patentable (*In re Best*, 562 F.2d 1252, 1254, 195 USPQ 430, 433 (CCPA 1977)). Hence, regarding the instant application, the discovery that SDF-1α induces the regeneration of endogenous cardiomyocytes, while interesting, does not render the claims patentable over Peterson and Hung et al. "Products of identical chemical composition can not have mutually exclusive properties." A chemical composition and its properties are inseparable. Therefore, if

the prior art teaches the identical chemical structure, the properties applicant discloses and/or claims are necessarily present. *In re Spada*, 911 F.2d 705, 709, 15 USPQ2d 1655, 1658 (Fed. Cir. 1990).

3. Claim 47 is rejected under 35 U.S.C. 103(a) as being unpatentable over Peterson, BE (US 2002/0094327; priority to 05 November 2000 and Hung et al. (US 2003/0171294; priority to 13 August 1999) as applied to claims 35, 37, 43, 46, 57 above, and further in view of Rempel et al. (Clin Can Res 6: 102-111, 2000). The basis for this rejection is set forth at pages 7-8 of the previous Office Action (03 February 2009).

Applicant's arguments (06 August 2009), as they pertain to the rejection of record have been fully considered but are not deemed to be persuasive for the following reasons.

Applicant argues that as discussed above, Peterson and Hung et al. do not suggest the claimed method because the combination of these cited art does not render as obvious a method of intramyocardial or intracoronary administration of SDF-1 effecting endogenous cardiomyocyte regeneration. Applicant asserts that it is not obvious to one skilled in the art at the time the invention was made to modify the method of intramyocardially or intracoronarily administering SDF-1α to heart tissue as allegedly taught by Peterson and Hung et al. by substituting SDF-1α with SDF-1β as taught by Rempel et al. Applicant argues that although it was known that SDF-1β existed at the time of Peterson, Peterson explicitly recites SDF-1α but never recites SDF-1β. Thus, one skilled in the art seeing Peterson only refers to SDF-1α would not immediately consider SDF-1α as replaceable with SDF-1β.

Applicant's arguments have been fully considered but are not found to be persuasive. The Examiner acknowledges that Peterson does not disclose utilization of SDF-1β. However, Rempel et al. teaches that the SDF-1 gene encodes two isoforms, SDF-1α and SDF-1β, that arise from alternative splicing (page 102, column 2, last paragraph). Rempel et al. also disclose that these isoforms differ only in that SDF-1β contains four additional 3' amino acids (page 102, column 2, last paragraph). Both isoforms interact with the same seven-transmembrane G protein-coupled receptor, CXCR4 (page 103, column 1, 1st full paragraph). Hence, SDF-1α and SDF-1β have the same function. Rempel et al. also disclose SDF1-deficient mice exhibit hematopoietic, cardiac, and cerebellar defects (page 103, column 1, bottom of 1st full paragraph). Therefore, it would have been obvious to the person of ordinary skill in the art at the time the invention was made to modify the method of intramyocardially or intracoronarily administering SDF-1α to heart tissue as taught by Peterson and Hung et al. by substituting SDF-1α with SDF-1β as taught by Rempel et al. Since Rempel et al. teach that SDF-1α and SDF-1β are isoforms encoded from the SDF-1 gene and that SDF-1β only contains four additional amino acids as compared to SDF-1α, one skilled in the art would have been motivated to substitute the utilization of SDF-1α for the SDF-1β to achieve the predictable result of treating a subject suffering from damaged heart tissue. Furthermore, MPEP 2144.09(I) states that a *prima facie* case of obviousness may be made when chemical compounds have very close structural similarities and similar utilities. "An obviousness rejection based on similarity in chemical structure and function entails the motivation of one skilled in the art to make a claimed compound, in the expectation that compounds similar in structure will have similar properties."

In re Payne, 606 F.2d 303, 313, 203 USPQ 245, 254 (CCPA 1979); *In re Papesch*, 315 F.2d 381, 137 USPQ 43 (CCPA 1963); *In re Dillon*, 919 F.2d 688, 16 USPQ2d 1897 (Fed. Cir. 1991).

4. Claims 49-51 are rejected under 35 U.S.C. 103(a) as being unpatentable over Peterson, BE (US 2002/0094327; priority to 05 November 2000) and Hung et al. (US 2003/0171294; priority to 13 August 1999) as applied to claims 35, 37, 43, 46, 57 above, and further in view of Isner et al. (WO 99/45775; 16 September 1999).

The teachings of Peterson and Hung et al. are set forth above.

Peterson and Hung et al. do not teach specific disorders of heart tissue.

Isner et al. teaches a method for increasing vascularization comprising administering to a mammal an effective amount of a vascularization modulating agent, such as stromal derived factor-1 (SDF-1) (bottom of page 4 through the top of page 5). Isner et al. disclose that the methods of the invention have a wide spectrum of uses in a human patient, i.e., use in the prevention or treatment of at least cerebrovascular ischemia, ischemic cardiopathy, and myocardial ischemia (page 15, lines 1-5). Isner et al. also teach that the ischemia may be especially adversely impact hear or brain tissue as often occurs in cardiovascular disease or stroke (page 15, lines 6-10). Isner et al. indicate that agents can be administered directly, i.e., intra-arterially (page 19, lines 17-18).

It would have been obvious to the person of ordinary skill in the art at the time the invention was made to modify the method of intramyocardially or intracoronarily administering SDF-1α to heart tissue as taught by Peterson and Hung et al. to treat disorders of heart tissue as taught by Isner et al. The person of ordinary skill in the art would have been motivated to make

that modification in order to localize cell migration/differentiation and tissue repair to the ischemic heart (see for example, Hung et al. page 1, [0007]; Peterson, page 1 [0004, 0006]). The person of ordinary skill in the art reasonably would have expected success because similar proteins and agents were already being intramyocardially and intracoronarily administered to the heart to treat ischemic diseases at the time the invention was made (see Hung et al.).

Conclusion

No claims are allowable.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Bridget E. Bunner whose telephone number is (571) 272-0881. The examiner can normally be reached on 9:00-5:30 M-F.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Manjunath Rao can be reached on (571) 272-0939. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

BEB
Art Unit 1647
09 September 2009

/Bridget E Bunner/
Primary Examiner, Art Unit 1647